"STUDY OF SEIZURES IN INTENSIVE MEDICAL CARE UNIT"

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CERTIFICATE

This is to certify that this dissertation entitled **"Study of seizures in intensive medical care unit"** submitted by **Dr. S.GANESAN** appearing for M.D. Branch I General Medicine Degree examination in April -2011 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India

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The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) inGeneral Medicine.

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LIST OF ABBREVATIONS

CNS	-	Central Nervous System
ICH	-	Intracerebral Hemorrhage
SAH	-	Sub Arachnoid Hemorrhage
EEG	-	Electro Encephalogram
IMCU	-	Intensive Medical Care Unit
ICU	-	Intensive Care Unit
AED	-	Anti Epileptic Drugs
PHT	-	Phenytoin
CBZ	-	Carbamazepine
VPA/VA	-	Valproic Acid
BDZ	-	Benzodiazepines
DZ	-	Diazepam
CVT	-	Cortical Vein Thrombosis
TBM	-	Tuberculous meningitis
SOL	-	Space Occupying Lesion
EPC	-	Epileptia Partialis Continua
CVA	-	Cerebrovascular Accident
CAD	-	Coronary Artery Disease
ACS	-	Acute Coronary Syndrome
CPGT	-	Complex partial with secondary generalization
CPS	-	Complex partial seizure
GTCS	-	Generalised tonic clonic seizures
SPGT	-	Simple partial with secondary generalization
SPS	-	Simple partial seizure
STE/SE	-	Status Epilepticus

TABLE OF CONTENTS

S.NO.	TITLE	Page no.
1	Introduction	1
2	Review of Literature	2
3	Aim of Study	30
4	Materials And Methods	31
5	Observations & Results	34
6	Discussion	53
7	Conclusion	59
8	Summary	60
9	Bibliography	
10	Proforma	
11	Master Chart	

INTRODUCTION

Seizures are devastating events in a person's life. Their very presence suggests that something is wrong with the Brain.

Seizures are relatively common neurological complication in patients admitted in intensive medical care unit. They commonly arise from comorbidities associated with the ICU experience. Most ICU seizures occur in patients who have not had a prior episode or for whom neurological pathology was part of the primary admitting diagnosis.

The ICU environment unfortunately presents unique challenges and difficulties with regard to the etiology, diagnosis and management of seizures, because in ICU, patients are critically ill, frequently with multiple organ dysfunction , treated with medications that may lower seizure threshold, and prevent good neurological estimation.

Since the incidence of seizures occurs most often in non-primary neurological patients, it is important for the general clinician, intensivist, and consulting neurologist to know about the potential for seizures in the ICU and to be aggressive in treating them.

Multiple seizure events or convulsive status epilepticus may lead to acidosis,hyperthermia,rhabdomyolysis,and trauma, and consequent high morbidity and mortality. Hence the need to diagnose and effectively treat seizure activity is imperative.

An effort has been made by this study to unravel the various aspects of seizures in IMCU in our hospital

REVIEW OF LITERATURE

Seizure (from the Latin Sacire, “to take possession of” is a paroxysmal event that occurs due to abnormal, excessive, hyper synchronous discharges from an aggregate of central nervous system (CNS) neurons[1, 2].

Historical Background

Seizures have been recognized since antiquity. One of the earliest descriptions[3] of a secondarily generalized tonic-clonic seizure was recorded over 3000 years ago in Mesopotamia, which was attributed to the God of the Moon. Epileptic seizures were described in ancient cultures including those of China, Egypt, and India.

Basic concepts surrounding seizures and epilepsy are found in Ancient Indian medicine which dates back to Vedic period of 4500-1500BC.In the Ayurvedic literature of Charaka Samhita, seizures were identified and epilepsy was described as *"apasmara"* which means *"loss of consciousness"*.[4]

The foundation of our modern understanding of the derangement of function seen in seizures and epilepsy was laid in the 19th century with the work of Hughlings Jackson. Working in Germany during the 1920s, Hans Berger, a psychiatrist, developed the human electroencephalograph. Another recent stimulus towards the understanding and treatment of seizures in the last few decades has been the development in

neuroimaging. Recent technology has revealed many of the more subtle brain lesions producing seizures.

Definitions of Terms

[As per the International League against Epilepsy (ILAE) Guidelines (Jallon, 1993; Roger, 1989).] [5]

A **nonepileptic event** is a clinical event presumed to be unrelated to abnormal and excessive neuronal discharge. An example of a nonepileptic event is syncope.

An **epileptic seizure** is a clinical event presumed to result from an abnormal and excessive neuronal discharge. The clinical symptoms are paroxysmal and may include impaired consciousness and motor, sensory, autonomic, or psychic events perceived by the subject or an observer.

A **provoked seizure** is an acute symptomatic seizure that occurs following a recent acute disorder such as a metabolic insult, toxic insult, CNS infection, stroke, brain trauma, cerebral hemorrhage, medication toxicity, alcohol withdrawal, or drug withdrawal.

An **unprovoked seizure** is a cryptogenic or a remote symptomatic seizure.

Epilepsy occurs when 2 or more epileptic seizures occur unprovoked by any immediately identifiable cause. The seizures must occur more than 24 hours apart. In epidemiologic studies, an episode of

status epilepticus is considered as a single seizure. Febrile seizures and neonatal seizures are excluded from this category.

Classification of Seizures

Seizures are divided into two broad categories--generalized and partial . Generalized seizures arise from both sides of the brain simultaneously. Partial (ie, focal) seizures occur within one or more restricted regions of the brain and are a secondary effect of a localized physiologic or structural abnormality of the brain (eg, tumor, dysplasia, stroke, trauma).

International League Against Epilepsy Revised Classification of Epileptic Seizures[1]

Classification of Seizures

1. Partial seizures

- Simple partial seizures (with motor, sensory, autonomic, or psychic signs)
- Complex partial seizures
- Partial seizures with secondary generalization

2. Primarily generalized seizures

- Absence (petit mal)
- Tonic-clonic (grand mal)
- Tonic
- Atonic
- Myoclonic

3. Unclassified seizures

- Neonatal seizures
- Infantile spasms

Classification of status epilepticus:

Status epilepticus refers to continuous seizures or repetitive, discrete seizures with impaired consciousness in the interictal period. A more practical definition is to consider status epilepticus as a situation in which the duration of seizures prompts the acute use of anticonvulsant therapy. For GCSE, this is typically when seizures last beyond 5 min.

Status epilepticus has numerous subtypes

1. Generalized convulsive status epilepticus (GCSE) (e.g., persistent, generalized electrographic seizures, coma, and tonic-clonic movements),
2. Nonconvulsive status epilepticus (e.g., persistent absence seizures or partial seizures, confusion or partially impaired consciousness, and minimal motor abnormalities)

Epidemiology:

In contemporary society, the frequency and importance of epilepsy can hardly be overstated. The *prevalence* of epilepsy is 4-8 per cent[6]. The *causes* of epilepsy differ in different parts of the world. Infections as a cause of epilepsy are more common in developing countries. Solitary lesions of neuro cysticercosis are an important cause of epilepsy in India.

From the epidemiologic studies of Hauser and colleagues, one may extrapolate an incidence of approximately 2 million individuals in the United States who are subject to epilepsy (i.e., chronically recurrent cerebral cortical seizures) and predict about 44 new cases per 100,000 population occur each year.

Over two-thirds of all epileptic seizures begin in childhood (most in the first year of life), and this is the age period when seizures assume the widest array of forms. The incidence increases again slightly after age 60. It is, however, notable that in striking contrast to the many treatments available for epilepsy, as pointed out by J. Engle, 80 to 90 percent of epileptics in the developing world never receive treatment.

A review by Bleck et al.noted that approximately 12% of patients admitted with a nonneurological primary diagnosis incurred neurological events during their critical illness. Of these incidence of seizures(28.1%) closely followed the metabolic encephalopathy as a leading neurological complication

Etiology of seizures in IMCU[7]:

1. Anoxic encephalopathy
2. CNS infections
    - Brain abscess
    - Encephalitis
    - Meningitis

- Subdural empyema

3. Cerebrovascular disorders
    - CNS vasculitis
    - Ischaemic stroke
    - Cortical vein thrombosis
    - Hemorrhage (ICH and SAH)
    - Lupus cerebritis
    - Thrombotic Thrombocytopenic Purpura
4. Drug intoxication[8]
    - Antibiotics with proconvulsant effects
    - High dose penicillin in patients with renal failure
    - Imipenem – cilastin in patients with renal failure or damaged BBB
    - Cocaine and other central stimulants
    - Dopamine antagonists
    - Meperidine metabolites
    - Theophyllin
    - Tramadol
    - Tri cyclic anti depressants
5. Drug / substance withdrawal
    - Alcohol
    - Benzodiazepines
    - Barbiturates

- Opiates
- Withdrawal of other anticonvulsants in patients with h/o seizure or a seizure disorder

6. Head trauma
   - Contusion
   - Hemorrhage
7. Metabolic encephalopathy- acute severe hypo osmolality (water intoxication)
8. Hepatic failure
9. Acute renal failure
10. Chronic renal failure
    - Drugs or metabolite intoxication
    - Dialysis dementia
11. Tumor
    - Primary
    - Metastatic
12. Hypertensive encephalopathy
13. Inflammatory diseases
    - Vasculitis
    - Acute disseminated encephalomyelitis

Pathophysiology of ICU Seizures[9] :

The manifestations of ictal process emanate from a few common cellular mechanisms and brain foci. The cerebral cortex is the nidus for most clinically evident seizures, and into that category also falls the hippocampus, the most rudimentary cortical element of the medial temporal lobe and thalamus.

As evidenced by the cortical EEG, the fundamental marker for an epileptic paroxysm is the interictal spike, which is the electrical finger print of the intracellular paroxysmal depolarizing shift (PDS) The interictal spike is generated by a synchronous firing of a network of local neurons, coupled with periods of inhibition via K+ current activated hyperpolarisation. Neurons in particular that are readily disposed to such activity are the pyramidal cells of cortical layer V and the CA 3 neurons in the hippocampus. The paroxysmal discharge may be induced by a local increase in excitation, decrease inhibitory neurotransmission, or alteration in Na^{+} or K^{+} current conductance. On a regional scale, ictal events may spread by disruption of the local chemical environment, such as alterations in K^{+} or Mg2+. Such spread is not very rapid and it is estimated to be at 50 – 200 mm/sec. Clinically important seizure propagation must clearly utilize synaptic transmission via networked connections.

Clinical Manifestations:

Determining the type of seizure that has occurred is essential for focusing the diagnostic approach on particular etiologies, selecting the appropriate therapy, and for providing vital information regarding prognosis. In 1981, the International League against Epilepsy (ILAE) published a modified version of the International Classification of Epileptic Seizures which has continued to be a useful classification system. This system is based on the clinical features of seizures and associated electroencephalographic findings.

A fundamental principle is that seizures may be either partial (synonymous with focal) or generalized. *Partial seizures* are those in which the seizure activity is restricted to discrete areas of the cerebral cortex. *Generalized seizures* involve diffuse regions of the brain simultaneously. Partial seizures are usually associated with structural abnormalities of the brain. In contrast, generalized seizures may result from cellular, biochemical, or structural abnormalities that have a more widespread distribution.

Three problems occur in recognizing the seizure in IMCU

1. Complex partial seizures in patients with already impaired consciousness
2. Seizures in patient receiving NMJ blockade

3. Misinterpretation of movement disorders and psychogenic disturbances as seizures

EEG is useful at this time in particular to diagnose non convulsive status epilepticus

Diagnostic Approach[10]:

The most important intervention done during a single seizure is careful observation. The first step in evaluating a suspected seizure is to determine whether the event was, in fact, a seizure .

Convulsive syncope occurs when there is severe or prolonged reduction of blood flow to the brain, resulting in an event resembling tonic clonic seizure.

Diagnosis is based on possible provocative factors in the medical history (eg, pain, dehydration), physical examination (eg, orthostatic blood pressure check), and studies such as electrocardiography and tilt table testing.

Another common imitator of epileptic seizure is the nonepileptic psychogenic seizure. No single feature reliably differentiates the two disorders. However, many ictal features of nonepileptic psychogenic seizure are uncommon in epileptic seizures. For example, such features as gradual onset, stopping and restarting of motor activity, out-of-phase clonic movements of the extremities, vocalization in the middle of the seizure rather than at the start, pelvic thrusting, and lack of body rigidity

are more common in psychogenic seizures than in tonic-clonic seizures. In addition, the typical duration of a tonic-clonic seizure is 50 to 92 seconds, whereas the range for psychogenic seizures is 20 to 805 seconds[11].

Furthermore, some epileptic seizures have symptoms that are frequently misdiagnosed as psychogenic. Frontal lobe complex partial seizures often last less than 1 minute and sometimes include rocking, kicking, "bicycling," pelvic thrusting, genital manipulation, and cursing. Their lack of postictal symptoms also makes frontal lobe seizures difficult to differentiate from psychogenic seizures.

Other NonEpileptic Paroxysmal Events

- Migraine (classic [with auras], basilar, confusional)
- Cerebrovascular event (transient ischemic attack)
- Periodic paralysis
- Sleep disorders (parasomnias, daytime amnestic episodes)
- Gastrointestinal disorders (reflux, motility disorders)
- Movement disorders (tics, Tourette's syndrome, nonepileptic myoclonus, paroxysmal choreoathetosis, shuddering attacks)
- Psychiatric disorders (panic, somatization, dissociation, conversion [nonepileptic psychogenic seizures])
- Drug toxicity and substance abuse
- Breath-holding spells

History of the event[10]

A description of the circumstances during a paroxysmal event can provide important diagnostic clues. A witnessed, 90-second episode that involved loss of consciousness, stiffening, and jerking of the extremities followed by muscle soreness, headache, and the need to sleep for several hours afterwards strongly suggests a tonic-clonic seizure

Key Elements in History

Before the event

- Unusual stress (eg, severe emotional trauma)
- Sleep deprivation
- Recent illness
- Unusual stimuli (eg, flickering lights)
- Use of medications and drugs
- Activity immediately before event (eg, change in posture, exercise)

During the event

- Symptoms at onset (eg, aura)
- Temporal mode of onset: gradual versus sudden
- Duration: brief (ictal phase <5 min) versus prolonged
- Stereotypy: duration and features of episodes nearly identical versus frequently changing
- Time of day: related to sleep or occuring on awakening
- Ability to talk and respond appropriately

- Ability to comprehend
- Ability to recall events during the seizure
- Abnormal movements of the eyes, mouth, face, head, arms, and legs
- Bowel or bladder incontinence
- Bodily injury

After the event

- Confusion
- Lethargy
- Abnormal speech
- Focal weakness or sensory loss (ie, Todd's paralysis)
- Headache, muscle soreness, or physical injury

Past medical history

A review of the events leading up to the seizure may reveal factors that suggest that it was provoked. Causes of provoked seizures include alcohol withdrawal, substance abuse, hypoxia, fever, electrolyte imbalance, hypoglycemia, and sleep deprivation.

Drug history

Theophylline, meperidine hydrochloride , isoniazid , antipsychotic drugs (especially clozapine and phenothiazines), radiocontrast dyes, alkylating agents, and ß-lactam antibiotics are among the most commonly implicated medications in seizure.

However, many other drugs can cause seizure, including lidocaine hydrochloride, general anesthetics, tricyclic antidepressants , selective serotonin reuptake inhibitors, bupropion hydrochloride, acyclovir, ßblockers, and decongestants (eg, phenylpropanolamine hydrochloride). Also, seizures can be provoked by alcohol withdrawal as well as use of cocaine.

Physical examination

A thorough physical examination can help to uncover possible causes of a seizure. Findings may include evidence of trauma, infection, malignancy, congenital anomalies, and prior neurologic events (eg, focal weakness, spasticity suggesting previous stroke).

During an emergency department evaluation of a patient immediately after a seizure, vital signs should be measured and a general medical examination performed. Guidelines for physical examination are as follows:

- Examine the patient for injuries from the seizure or a fall.
- Check oxygen saturation and auscultate the chest for possible aspiration.
- Measure heart rhythm and rate, blood pressure, and orthostatic changes for assessment of syncope.
- Auscultate for carotid murmurs or carotid bruits and sources of embolic stroke.

- Check for rapid pulses, which are often present after seizure and may help in evaluation of psychogenic seizures.

An electrocardiogram should be obtained to identify cardiac rhythm, detect possible ischemia, and to measure the QT interval. Prolonged QT syndrome often presents with simple or convulsive syncope. Electrocardiography and 24-hour ambulatory continuous electrocardiographic (Holter) monitoring can help identify cardiac arrhythmias. The possibility of a recent myocardial infarction should be considered, particularly in elderly patients, in whom myocardial infarction may occur from the stress of a seizure.

Neurologic examination

The purpose of the neurologic examination is to identify focal or diffuse cerebral dysfunction. This information is particularly helpful in localization related epilepsy. The presence of various features offers clues to the focus of a seizure. For example, aphasia suggests a left frontal, temporal, or parietal onset. Right or left hemiparesis suggests foci from the contralateral motor cortex.

In initial evaluation of a seizure, patients should be observed for fluency of language, facial asymmetry, gaze preferences, and pupillary asymmetry. The last presents in patients who have herniation from brain swelling caused by parenchymal or epidural bleeding and in those who have a rapidly growing brain tumor. The presence of pronator

drift may indicate subtle weakness not detected by strength testing. Sensory deficits suggest parietal lobe dysfunction. An extensor plantar response may be noted for some time after a seizure and is not necessarily a pathologic finding.

Diagnostic testing

The investigations are aimed to obtain lab studies based upon age and etiology, basic investigations, toxicology screening and anticonvulsant levels (if indicated by H/o ingestion or existent therapy).

Laboratory workup is an essential part of evaluation of seizure. Measurement of glucose, calcium, magnesium, thyroid hormone, and liver enzyme levels, as well as toxicology screening (including blood alcohol levels), may reveal common medical causes of seizures. A complete blood cell count may suggest infection, anemia, or sickle cell disease. In patients suspected to have had an infection or a fever or to have exhibited abnormal behavior just before the event, lumbar puncture should be performed after assessment of the possible risks of the procedure (eg, coagulopathy, mass lesion). Patients who are immunocompromised because of corticosteroid use, recent transplantation, or HIV infection should undergo cerebrospinal fluid evaluation to detect possible fungal, bacterial, or viral infection. In patients with a systemic malignant condition, cytologic evaluation of cerebrospinal fluid can identify meningeal carcinoma.

Electroenchephologram:

- EEG should be performed within 24 hours of the seizure because it is significantly more sensitive when obtained during that period (King,1998). If the routine EEG findings are normal, a sleep-deprived EEG should be performed.
- Standard EEG detects epileptiform discharges in 29% of patients. Standard EEG combined with sleep-deprived EEG shows epileptiform discharges in 48% of patients (van Donselaar, 1992)[12].
- In 2000, Simpson et al described a case in which the placement of an insertable loop recorder, an important new tool in the diagnostic evaluation of patients with syncope, led to an unexpected diagnosis of a seizure. Whenever cardiovascular causes are considered as the cause of a patient's spells but cannot be proven with conventional investigations, the use of the insertable loop recorder should be considered.
- Schreiner and Pohlman-Eden studied the value of an EEG taken within 48 hours of the first seizure in an adult. They found that 38.0% of patients without seizure recurrence had normal EEGs, while only 10.2% of patients with seizure recurrence had normal EEGs. Focal epileptiform activities were found significantly more frequently(26.5% vs. 13.0%) in patients with seizure recurrence than in patients without seizure recurrence.

Limitation of EEG[13]:

An estimated 0.4% of adults and 2.8% of children who have never had a seizure may have interictal epileptiform discharges . Furthermore, a normal EEG does not refute the diagnosis of epilepsy. The initial EEG reveals epileptiform activity in only 40% of the patients with probable epilepsy.

Imaging studies

The role of imaging studies depends on the stage of evaluation. Immediately after a seizure, computed tomography can detect the presence of bleeding or gross structural lesions. However, magnetic resonance imaging is the study of choice because it is more sensitive and specific for evaluating structural lesions of brain parenchyma. Particular attention should be directed to the hippocampus for evaluation of lesions (eg, mesial temporal sclerosis) and the cortical architecture for detection of abnormalities (eg, dysplasia).

Principles of Seizure Management[1]:

Therapy for a patient with a seizure disorder is almost always multimodal and includes treatment of underlying conditions that cause or contribute to the seizures, avoidance of precipitating factors, suppression of recurrent seizures by prophylactic therapy with antiepileptic medications or surgery, and addressing a variety of psychological and social issues.

Treatment of Underlying Conditions

If the sole cause of a seizure is a metabolic disturbance such as an abnormality of serum electrolytes or glucose, then treatment is aimed at reversing the metabolic problem and preventing its recurrence. Therapy with antiepileptic drugs is usually unnecessary unless the metabolic disorder cannot be corrected promptly and the patient is at risk of having further seizures. If the apparent cause of a seizure was a medication (e.g., theophylline) or illicit drug use (e.g., cocaine), then appropriate therapy is avoidance of the drug; there is usually no need for antiepileptic medications unless subsequent seizures occur in the absence of these precipitants.

Seizures caused by a structural CNS lesion such as a brain tumor, vascular malformation, or brain abscess may not recur after appropriate treatment of the underlying lesion. However, despite removal of the structural lesion, there is a risk that the seizure focus will remain in the surrounding tissue or develop *de novo* as a result of gliosis and other processes induced by surgery, radiation, or other therapies. Most patients are therefore maintained on an antiepileptic medication for at least 1 year, and an attempt is made to withdraw medications only if the patient has been completely seizure-free. If seizures are refractory to medication, the patient may benefit from surgical removal of the epileptic brain region.

Avoidance and treatment of Predisposing Factors :

In the intensive care unit setting, seizures are a common neurological complication in both medical and post surgical patients. They commonly arise from the comorbities associated with the ICU experience.

Complications of Critical Illness Increasing Seizure Predisposition[7]:

- Hypoxia / ischemia
- Drug / substance toxicity
  - Antibiotics
  - Antidepressants
  - Antipsychotics
  - Bronchodilators
  - Local anesthetics
  - Immunosuppressives
  - Cocaine
  - Amphetamines
  - Phencyclidine
- Drug / substance withdrawal
  - Barbiturates
  - Benzodiazepines
  - Opiods
  - Alcohol

- Infection and fever
- Metabolic abnormalities
  - Hypophosphatemia
  - Hypocalcemia
  - Hypoglycemia
  - Renal / hepatic dysfunction
- Surgical injury (craniotomy)

Hence avoidance or correction of the above factors is essential to prevent the recurrent episodes of seizures in the IMCU settings.

Treatment

Optimal treatment of seizures in the ICU, involve both the acute cessation of ictal activity and preventing recurrence, ideally by removing or correcting the physiological trigger and providing pharmacological prophylaxis against recurrence. Most commonly, seizures manifest as single episodes which serve to alert the care givers in dramatic fashion that a metabolic or structural abnormality exists,

For immediate treatment of seizures, benzodiazepines are considered as the first line of therapy. They penetrate into brain rapidly, are potent GABA agonists, and serve to improve local inhibition of signal transmission. Commonly used benzodiazepines include diazepam, midazolam, or lorazepam. Each has a unique pharmacokinetic profile. Diazepam has been the popular standard, although its use against seizures

is waning owing to the superior properties of the other two, highly lipophilic, diazepam rapidly redistributes away from the serum into fat. The result is that its effective anticonvulsant duration is on the order of only a few minutes, although its elimination time from the body is many hours and the longest of the three agents (23). Such kinetics could possibly result in brief seizure control yet a prolonged sedative effect if large dosages are required.

Midazolam is also highly lipophilic and short acting, but is cleared by the liver much more rapidly than diazepam(> lOx) resulting in better correlation between drug effect and clearance (24). Lorazepam, a compound with greater water solubility that prolongs its serum half-life, is clinically effective for several hours (25). In a recent randomized controlled trial with patients in status epilepticus, lorazepam was found to be superior than diazepam or phenytoin alone in terminating clinical and EEG seizures (25). Thus, it is reasonable to use lorazepam to the treatment of all GTCS in the ICU since rapid, maximal control is desired. In patients in whom prolonged sedation may seriously confound neurological management, initial treatment of seizures may be instituted with a short acting benzodiazepine to be followed immediately by a loading dose of phenytoin.

Once seizures are controlled, monotherapy with phenytoin for seizures is advocated to lessen the complications of drug interactions.

Seizure recurrence should be managed first with acute treatment, again typically with benzodiazepines, followed by increasing serum concentrations of the primary anticonvulsant to high or maximal therapeutic levels. Should seizures become refractory, continuous EEG monitoring, intravenous phenytoin or inhalational anesthetics are recommended.

As alluded to earlier, it is important to realize that seizures occurring in the ICU setting may have unusual causes with complex features and treatments. Convulsions from theophylline toxicity carry a risk of morbidity and mortality that may exceed 40% (26) and part of the reason may be due to the fact that these seizures can be refractory to conventional anticonvulsant regimens (27). Repetitive seizures and status epilepticus may result. Hemoperfusion, dialysis, and activated charcoal all have their advocates for acute therapy, and some experts believe that aggressive measures should be initiated if theophylline serum levels reach 100 μg/ml (35,36,37).Isoniazid is another drug that requires non-conventional therapeutics. Due to its action as an antagonist to pyridoxal phosphate, treatment includes intravenous pyridoxine (27,28).

Treatment of other drug-induced seizures generally respond to benzodiazepines or barbiturates, and these should be considered first line options (29). Phenytoin is not particularly effective against most drug-induced convulsions, especially those triggered by B-lactam antibiotics.

Hemodialysis may be taken into consideration if seizures are recurrent, particularly if renal failure complicates drug elimination.

Treatment Protocol

Management of IMCU seizures and SE should include emergent medical management, termination of seizures, prevention of recurrence of seizures, and prevention or treatment of complications. Clinical assessment was used to monitor the response to the treatment in almost half the cases.

The specialized protocol for the treatment of seizures in IMCU setting is as follows.

As a general rule, one brief GTCS seizures in the ICU is not an indication for AED administration. By the time the drugs are available at the bedside, the seizure was usually over. Close monitoring of the patients vital signs, bed padding, and the initiation of a workup for identifying the cause are usually adequate measures.

Table : (A) MANAGEMENT OF BRIEF SINGLE ICU SEIZURE (<60S)[38]
Observe. Eliminate etiology. Consider chronic therapy: PHT (15-20mg/kg) or fosphenytoin (15-20mg/kg) PHT equivalents (PE) loading dose and 300-400mg/d. Goal serum of 10-20 microgram/ml or free level 1-2 microgram/ml. PHT intolerant patient: intravenous / oral VPA (15-20 mg/kg load, maintenance 600-3000mg/d) or oral CBZ (600-1200 mg/d). Seizure precautions: padded bed rails, increased observation.

Table : (B) MANAGEMENT OF A PROLONGED SEIZURE OR MORE THAN ONE SEIZURE IN THE ICU[38]
Check oxygen saturation, vital signs. Consider intubation, if risk of aspiration. Intravenous BDZ, e.g., lorazepam (1-2mg), DZ (10-20mg), or midazolam (2-5mg) with concurrent loading dose of PHT or fosphenytoin (PE) of 15-20 mg/kg and maintenance of 300-400 mg/d. PHT-intolerant patients: VPA (intravenous load 15-20mg/kg), with maintenance at 600mg every 6 hr. Seizure precautions: padded bed rails, increased observation.

Table : (C) MANAGEMENT OF STATUS EPILEPTICUS

ABC: preserve airway and oxygenation by intubation.

Measure blood glucose at bedside. Give 100 mg iv thiamine and glucose, only if <40-60 mg/100 dL or unable to have a fast result. At the same time draw blood for blood count, electrolytes, liver enzymes, creatinine kinase, toxicology screen, arterial blood gases, and AED levels.

Immediate intravenous BDZS: lorazepam (5-10mg), DZ (20-40mg), or midazolam (5-20 mg) over 5 min.

PHT loading dose of 20 mg/kg at 50 mg/min or fosphenytoin, 20 mg/kg PE at 150 mg/min. Consider VPA intravenous load of 15-20 mg/kg, maintenance 400-600 mg every 6 h in PHT-intolerant patients.

Consider EEG, if available

If seizure continue, PHT or fosphenytoin (additional 5-10 mg/kg or 5-10 mg/kg PE). Consider VPA intravenous load of 15-20 mg/kg, maintenance 400-600mg every hr.

Hemodynamic support consists of fluids, pressors, and inotropes.

Add more BDZ if necessary, and consider weaning infusion agent several hours later (preferably 12-24 h) while optimal serum anticonvulsant levels are documented.

If seizures persist, consider prolonged barbiturate or anesthetic coma with pentobarbital (12mg/kg at 0.2-0.4mg/kg/min followed by an infusion of 0.25-2 mg/kg/h) for continued EEG suppression.

(Ref-39,40,41,42,43,44,45,46,47,48,38)

Prophylactic Therapy for Seizures

Patients in the ICU frequently suffer from a cerebral disturbance which carries a risk of seizures. Thus the issue is raised whether the benefit of seizure prevention outweighs its potential toxicity. As stated earlier, many seizures in the ICU are manifestations of transient metabolic or physiological abnormalities and the risk of recurrence is low if homeostasis is restored. For patients with structural pathology of the CNS, however, the risk of recurrence is frequently high.

Cerebrovascular accidents are a precipitating cause of seizures in 3-6% of patients (30, 31). Whether all stroke patients, as a group, should receive prophylactic anticonvulsant therapy has not been fully established. Hemorrhagic stroke carries a two-fold increase in the risk of seizures compared to ischemic infarction. Thus elderly patients, who are confused at the beginning and those who do have an early seizure (<1 week after stroke) have a high risk of recurrence and should therefore be considered for prophylactic therapy (31).

Patients with an intracranial tumor are at high risk for seizures, and are frequently prescribed prophylactic seizure medication. It is known for certain that these patients with cerebral tumors, particularly gliomas, have a high recurrence rate of seizures. Once they occur, prophylactic therapy is mandated, although incompletely effective in more than 25% of cases (32).

In case of head injury, as stated previously, the risk of seizures in early post-trauma period is approximately 4%. Offered appropriate treatment, reports suggest that the occurrence of a seizure does not influence the ultimate neurological recovery (33). Prophylaxis against seizures has not been demonstrated to prevent the possibility of epilepsy following recovery in a prospective cohort. However, the likelihood of further seizures and neurological deterioration does support aggressive treatment following an initial convulsion (34).

Summary

Seizures occurring in the IMCU settings are more difficult to prevent, diagnose, and treat effectively than those manifesting outside the ICU or hospital. Diagnosis is challenging when only subtle clinical evidence is present and the EEG is not conclusive. The use of variety of medications and the clinical management in an ICU all tend to lower seizure threshold rather than elevate it . Moreover, treatment of seizures in an ICU setting can also be challenging even in the absence of status epilepticus[7] .

AIMS OF THE STUDY

1. To study the cause of seizures in patients admitted to the IMCU
2. To study the semiology of seizure and the relation of semiology to aetiology
3. To study the relation of seizures to the final outcome

MATERIALS AND METHODS

The study was done in Intensive medical care unit, Department of Medicine, Tirunelveli medical college, Tirunelveli. The study was done in association with the Departments of Neurology, Biochemistry, Pathology, Radiology and Microbiology.

The study was prospective cross sectional study designed to analyze the seizures in the patients admitted in IMCU. The sample size was 60 and the study period was from October 2009 to November 2010.

Case Selection:

1. All patients admitted to IMCU with seizure were taken for study
2. All patients who developed seizure while in IMCU treatment were taken for study
3. Only patient above the age of 12 were taken for study
4. Patients with psychogenic and pseudo seizure were excluded from the study

Methodology

Clinical data was collected from patients and witnesses in a systematic manner and added to a database, which included a checklist of seizure antecedents and the symptoms associated with seizure. The first task was to ascertain if at all, the presenting complaint was a seizure. In a few instances, even when the presenting history was ambiguous, seizure recurrences were witnessed for confirmation. The detailed history for all

patients was obtained and the **history regarding semiology** of seizure was taken. The involvement of the extremities, eye movements, flexion, extension, stiffening of the extremities including any focal movements were noted. The clinical diagnosis of the seizure type, whether partial or generalized was made. Significant past medical history if any was noted. Any evidence of post ictal neurological deficits were sought. The following illness were noted

1. Fever
2. Intercurrent illness
3. Head injury
4. Intoxication
5. Toxin exposure
6. CVA
7. Electrolyte imbalance

Significant past history regarding the seizure disorder, non compliance of the drugs, diabetes, hypertension, tuberculosis, HIV, SLE, COPD and bronchial asthma were noted.

Complete general examination of the patient and central nervous system examination was done. Routine blood investigations, urine analysis, blood sugar, urea, serum creatinine, sodium, potassium, chloride, bicarbonate, calcium, and magnesium levels estimation were done in all patients.

CT scan brain study plain and if needed contrast was done in most of the patients except in few patients in whom the general condition was so bad that patient could not be shifted for CT scan

In needed cases, MRI brain, MRA, MRV was also done

An attempt was made to do EEG in most of the patients with 16 channel digital EEG at the earliest.

OBSERVATION AND RESULTS

60 patients with seizures were studied. The clinical study was statistically analyzed and interpreted according to the appropriate tests of significance. The difference between various characters of patients was analyzed by Means and interpreted by students unpaired t' test. The significance of associations between the factors were analyzed and interpreted by the test of significance namely Chi' – square test (ψ^2). The p' value < 0.05 was taken into the account of the significance.

Table 1:

Sex distribution of patients

Sex	No	Percentage (%)
Male	39	65%
Female	21	35%

Among the total 60 study subjects ,39(65%) were male and remaining 21(35%) were female .the male to female ratio in this study was 1.8:1

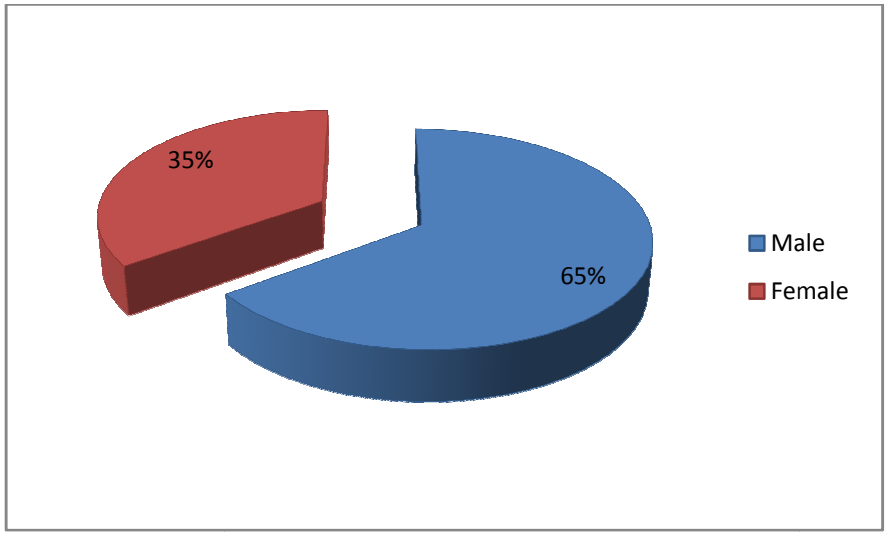


Table 2 :

Age related sex distribution.

Age group (years)	Male		Female		Total	
	No.	%	No.	%	No.	%
10-19	3	7.7	3	14.3	6	10
20-29	7	17.9	9	42.9	16	26.7
30-39	7	17.9	1	4.8	8	13.3
40-49	7	17.9	4	19	11	18.3
50-59	7	17.9	2	9.5	9	15
60-69	3	7.7	2	9.5	5	8.3
70-79	2	5.1	0	0	2	3.4
80-89	3	7.7	0	0	3	5.0
Total	39	100	21	100	60	100
Median(Range)	44(13-89)		28(13-65)		39.5(13-89)	
Mean± S.D	43.9±20		33.3±16.2		40.1±19.3	
't'	2.047.d.f.= 58					
Significance	P<0.05					

The above table 2 reveals that the ages of the study subject range from 13-89 and the median age of the study group was 39.5 years. The ages of the male patients range from 13-89 with median age as 44 and the ages of the female patients were ranged between 13-65 with the median age of 28 years. The mean ages of the males and females were 43.9±20 years, and 33.3±16.2 years respectively. The mean age difference between the males and female patients is statistically significant as P

value is less than 0.05 indicating that more females were admitted at younger age with seizure in this study.

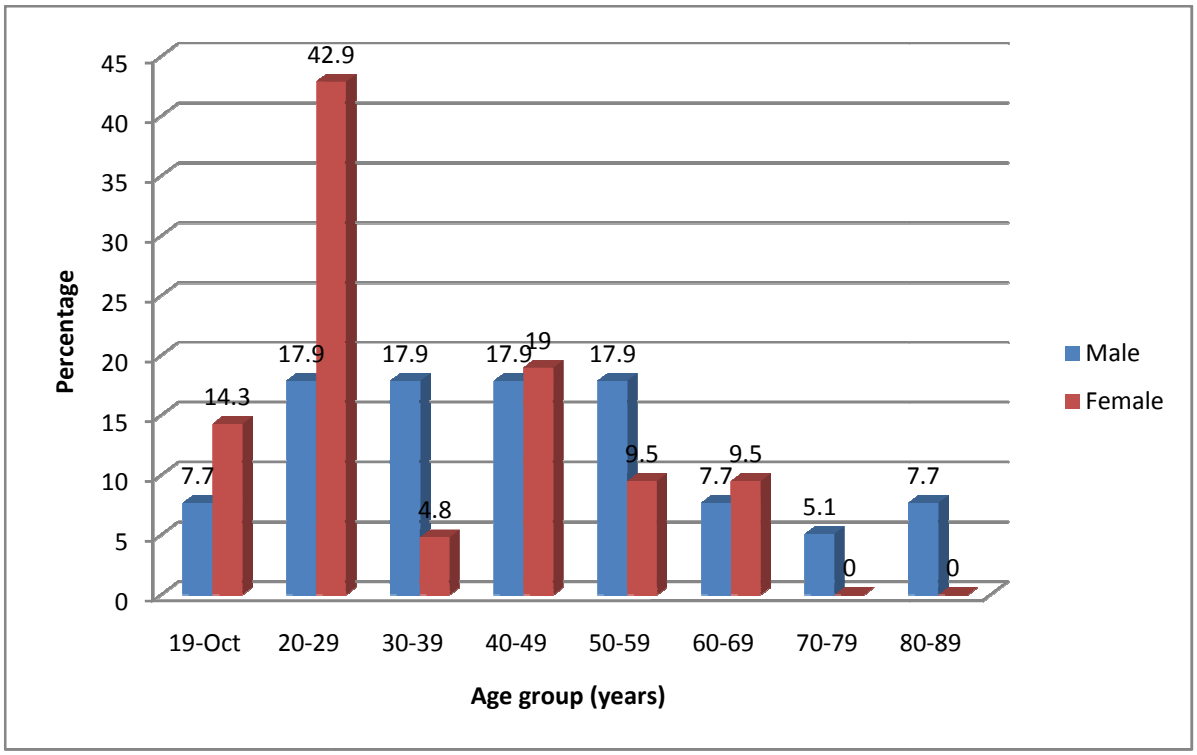


Table 3:

Patients with new onset seizure Vs known epileptic patient

Type	No	Percentage
New onset	36	60%
Known epileptic	24	40%

Analysis of the above table indicates that 60% of patients in the study developed new onset seizure and the remaining 40% are known epileptic who had recurrence of seizure

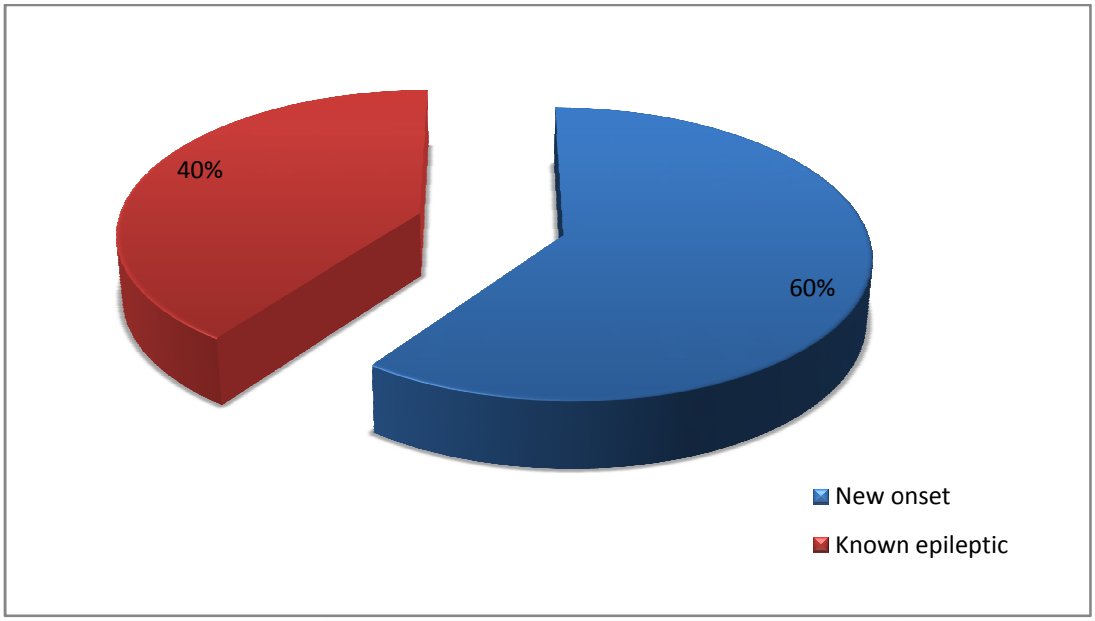


Table 4:

Age distribution of known epileptic and new onset seizure disorder patients

Age group	Known seizure patients		New onset seizure	
	No.	**%**	**No.**	**%**
10-19	4	16.7	2	5.6
20-29	5	20.8	11	30.6
30-39	4	16.7	4	11.1
40-49	4	16.7	7	19.4
50-59	4	16.7	5	3.9
60-69	3	12.5	2	5.6
70-79	0	0	2	5.6
80-89	0	0	3	8.3
Total	24	100	36	100
Mean± S.D	37.3±16.3		42.5±21	
't'	1.034			
Significance	P>0.05			

Age does not have a bearing on the recurrence of seizure in a known epileptic or for new occurrence of seizure in general public

Table 5:

Aetiology of seizure in known epileptic patients

Etiology	No	Percentage
Drug withdrawal	20	83.3%
Vascular		
Hemorrhage	2	8.3%
Infarct	1	4.2%
Metabolic		
Hyperglycemia	1	4.2%
Total	**24**	**100%**

The drug withdrawal (seen in 20 cases 83.3%) is the most common cause for seizure episode in the known epileptic patient. Among the 24 cases 3(12.5%) cases were due to vascular causes of which it was hemorrhage in 2 cases and infarct in one cases. Only one patient had recurrence of episode due to hyperglycemia.

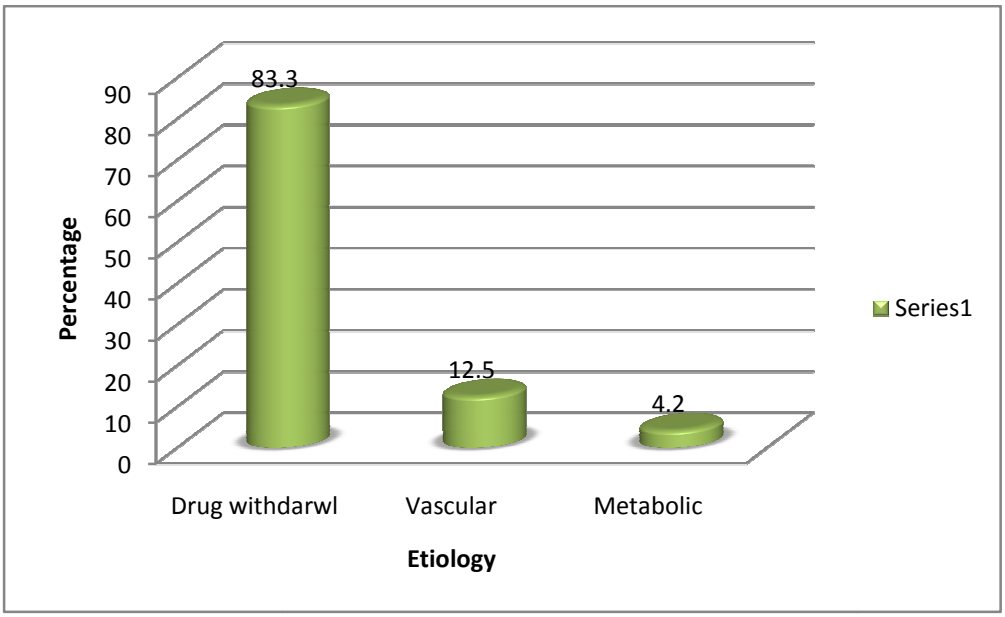


Table 6:

Causes of new onset seizure

Causes	No	Percentage
1.Vascular		
Infarct	8	22.2% } 25%
Hemorrhage +CVT	1	2.7%
2.Metabolic		
Hyperglycemia	4	11.1%
Hypoglycemia	2	5.5% } 25%
Hyponatremia	2	5.5%
Uremia	1	2.7%
3.Infection		
TBM	3	8.3%
Encephalitis	2	5.5%
4.Tumor/SOL	5	13.8%
5.Hypoxia	4	11.1%
6.Toxin	3	8.3%
7.Unidentified	1	2.7%
TOTAL	**36**	**100%**

Etiologies for the total 36 cases of new onset seizure were analyzed. Vascular and metabolic causes being the dominant cause for new onset seizure, each attributes about 25%. Infarct is the most common vascular etiology. Hyperglycemia is major metabolic abnormality causing seizure. Others are hypoglycemia, hyponatremia, and uremia.

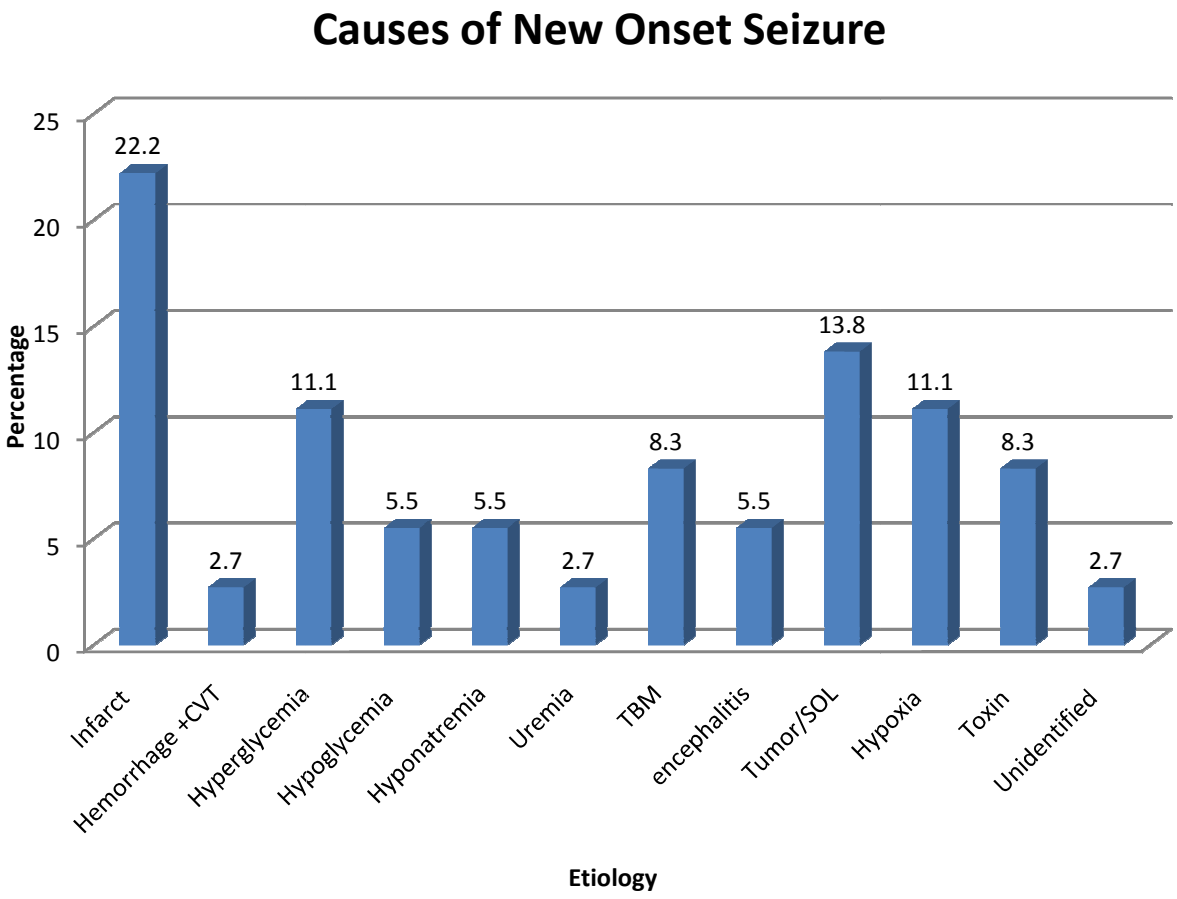


Table 7:

Type of seizure encountered

Type of seizure	Frequency	%
CPGT	6	10.0
CPS	3	5.0
GTCS	38	63.3
Myoclonus	1	1.7
SPGT	6	10.0
SPS	6	10.0
Total	**60**	**100**

The above table-7 evaluates the seizure types in the study group. GTCS type of seizure was seen in maximum no. of study cases. It is seen in 38 (63.3%) patients.

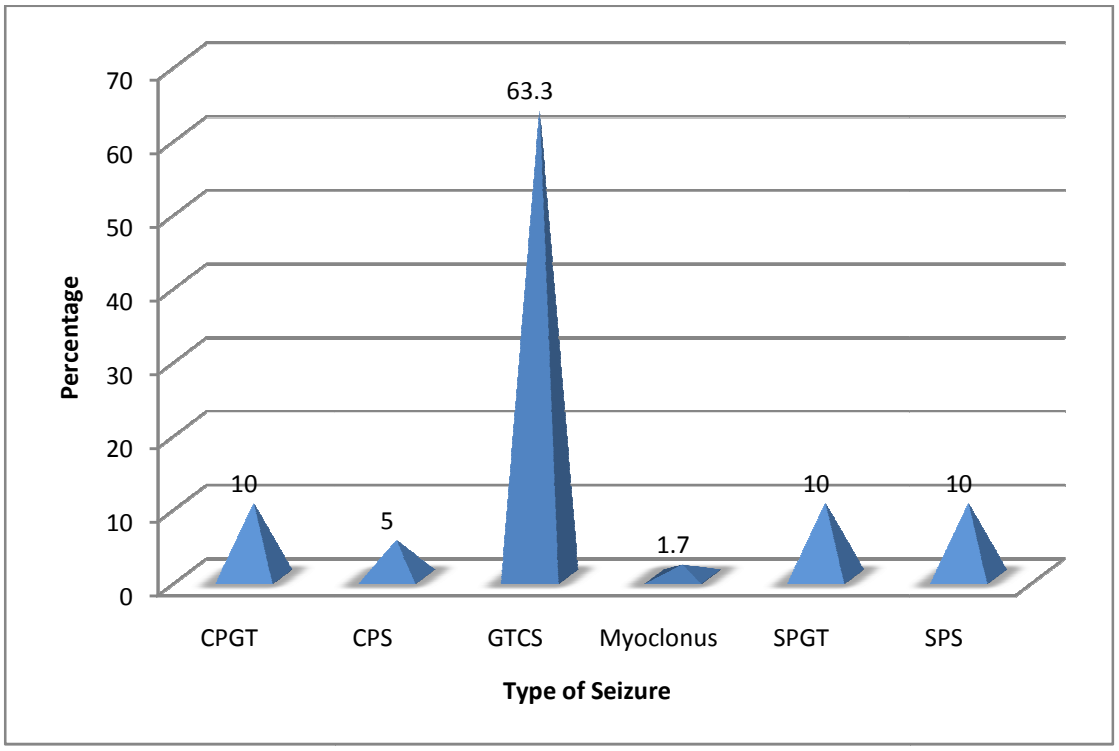


Table 8:

Irrespective of the age the study subjects were classified into two categories namely <median and median and above age.

Pattern of seizure according to the age group.

Type of seizure	AGE				Total	
	<Median		Median and above			
	Fre	%	Fre	%	Fre	%
CPGT	3	10.0	3	10.0	6	10.0
CPS	1	3.3	2	6.7	3	5.0
GTCS	21	70.0	17	56.7	38	63.3
Myoclonus	1	3.3	0	0.0	1	1.7
SPGT	2	6.7	4	13.3	6	10.0
SPS	2	6.7	4	13.3	6	10.0
Total	30	100	30	100	60	100

The above table 6, shows that GTCS was seen in maximum no. of patients(38i.e. 63.3%). Among the 38 cases 21(71%) were less than 40 years and 17(56.7%) cases were above 40 years. But the difference in the type of seizure according to the age group variation is not statistically significant (P value 0.686).

Table 9:

Causes of status epilepticus

Etiology	**No**	**Type**	**Percentage**
1.Known epileptic			
Drug withdrawal	6	GTCS	31.6%
2.New onset seizure			
Metabolic			
➢ Hyperglycemia	3	EPC	15.8%
➢ Hypoglycemia	1	GTCS	5.3%
Vascular			
➢ Infarct	1	CPGT	5.3%
Infection			
➢ Meningo encephalitis	1	GTCS	5.3%
Toxin	3	GTCS	15.8%
Tumor	1	GTCS	5.3%
Hypoxia	3	GTCS	15.8%
TOTAL	19		100%

Among the sixty cases, 19 patients(31.67%) had status epilepticus. The presence of status epilepticus among the seizure patients according to

etiology was associated in the above table -9. Drug withdrawal was the commonest cause for the status epilepticus as 6(31.6%) out of 19(100%) cases were due to it. Hyperglycemia most commonly present as epilepsy partialis continua(15.8%)

Causes of status epilepticus

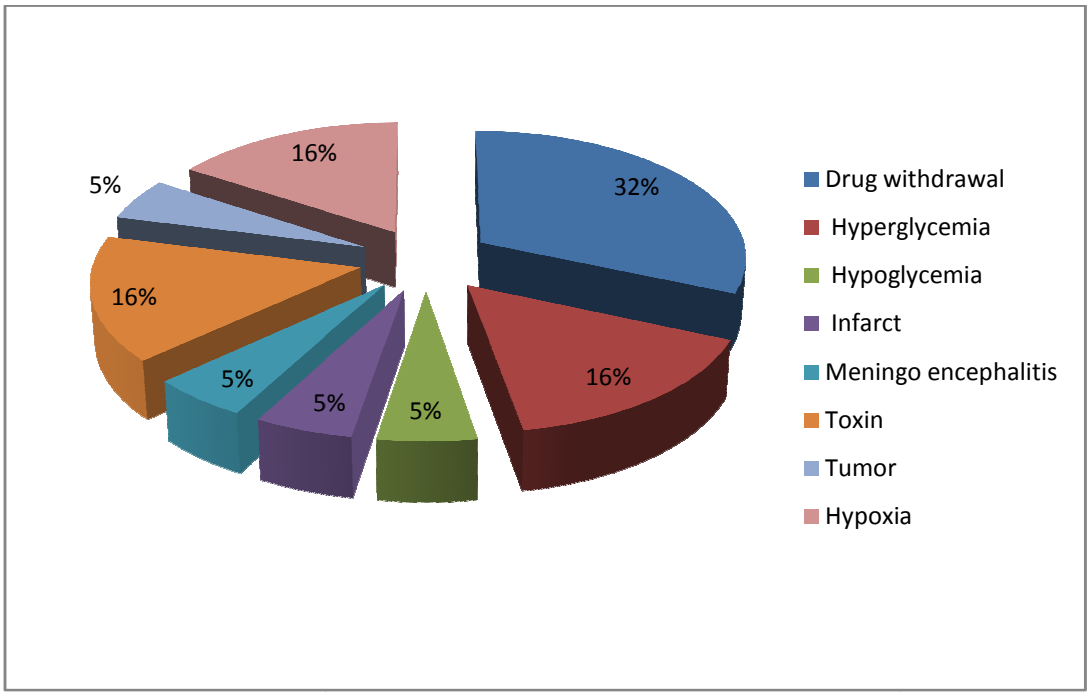


Table 10:

Etiology of status epilepticus with mortality

Etiology	**No**	**Discharged**	**Expired**
1.Known epileptic			
➢ Drug withdrawal	6	5	1
2.New onset seizure			
Metabolic			
➢ Hyperglycemia	3	2	1
➢ Hypoglycemia	1	1	0
Vascular			
➢ Infarct	1	1	0
Infection			
➢ Meningo encephalitis	1	1	0
Toxin	3	2	1
Tumor	1	2	0
Hypoxia	3	2	1
TOTAL	**19**	**15**	**4**

Among the 60 study patients 9 patients expired during the study. Out of the 9 (15.0%) mortality cases 4(21.1%) had status epilepticus.The above table reveals that the outcome of patients with status epilepticus depends upon the underlying etiology rather than the status itself.

Table 11:

Etiological association of type of seizure

Etiology	**Type of seizure**						**total**
Known epileptic	**CPGT**	**CPS**	**GTCS**	**MYOCLO**	**SPGT**	**SPS**	
Drug withdrawal	2	2	12	1	2	1	20
Vascular							
Hemorrhage	-	-	1	-	1	-	2
Infarct	-	-	1	-	-	-	1
Hyperglycemia	-	-	1	-	-	-	1
New onset seizure							
Vascular							
Infarct -acute	-	1	-	-	-	-	1
Chronic	1		1		2	2	6
Hemorrhage	1				1		2
Metabolic							
Hyperglycemia	-	-	1	-	-	3	4
Hypoglycemia	-	-	3	-	-	-	3
Hyponatremia	-	-	1	-	-	-	1
Uremia	-	-	1	-	-	-	1
Infection							
a.TBM	1	-	2	-	-	-	3
b.Meningoencephalitis	-	-	3	-	-	-	3
Tumor							
a.Primary	-	-	1	-	-	-	1
b.Secondary	1		2	-	-	-	3
Toxins							
a.Alcohol	-	-	2	-	-	-	2

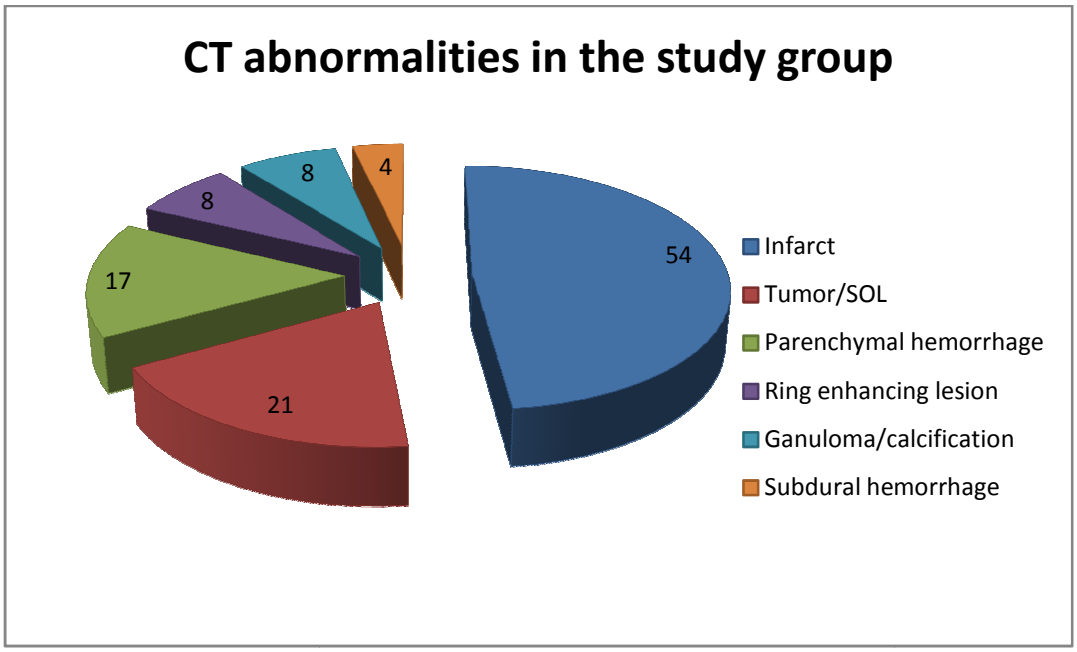
b.Endosulphan	-	-	1	-	-		1
Hypoxia							
a.Hanging	-	-	3	-	-	-	3
b.Pneumothorax	-	-	1	-	-	-	1
Unidentified	-	-	1	-	-	-	1
TOTAL	**6**	**3**	**38**	**1**	**6**	**6**	**60**

The above table list out the diagnosis of all the study subjects with underlying etiology that provoked seizure. The most common etiology is drug withdrawal (20cases). And the next common is vascular etiology (12 cases). In all the etiological diagnosis, GTCS type of seizure was the most common type.

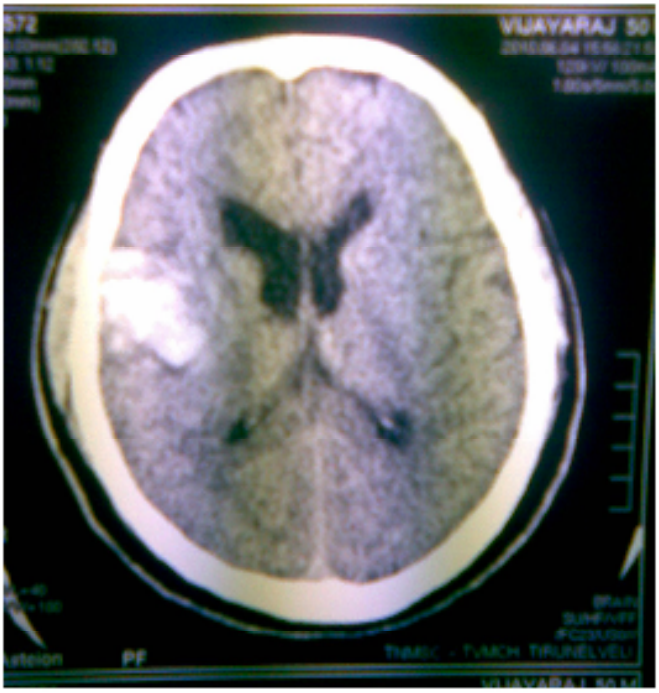
Table 12: Shows CT abnormalities in the study group

CT findings	No (%)
Infarct	13(54%)
Tumor/SOL	5(21%)
Parenchymal hemorrhage	4(17%)
Ring enhancing lesion	2(8%)
Granuloma/calcification	2(8%)
Subdural hemorrhage	1(4%)

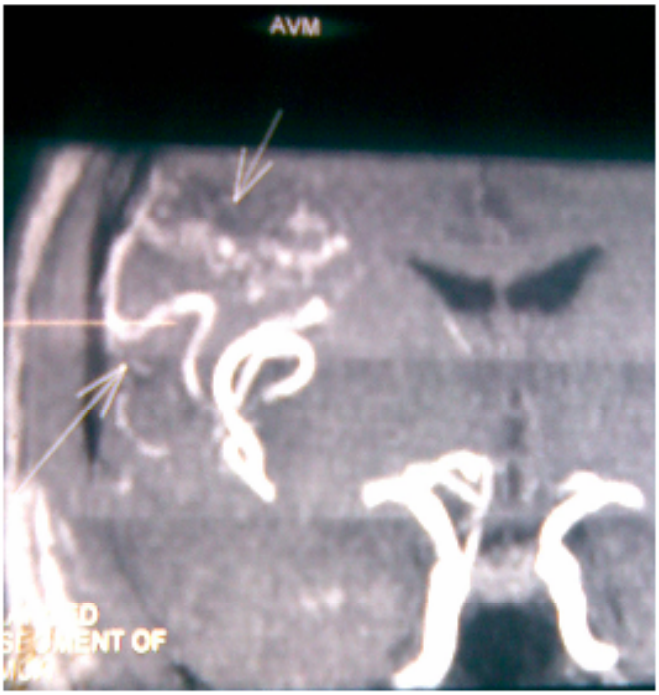
Cortical atrophy was found in combinations with various other findings. Chronic infarct with gliosis is seen in maximum of number of patients.



INTRACEREBRAL HEMORRHAGE



MRA showing Arterio Venous Malformation



MRI showing Large Meningioma

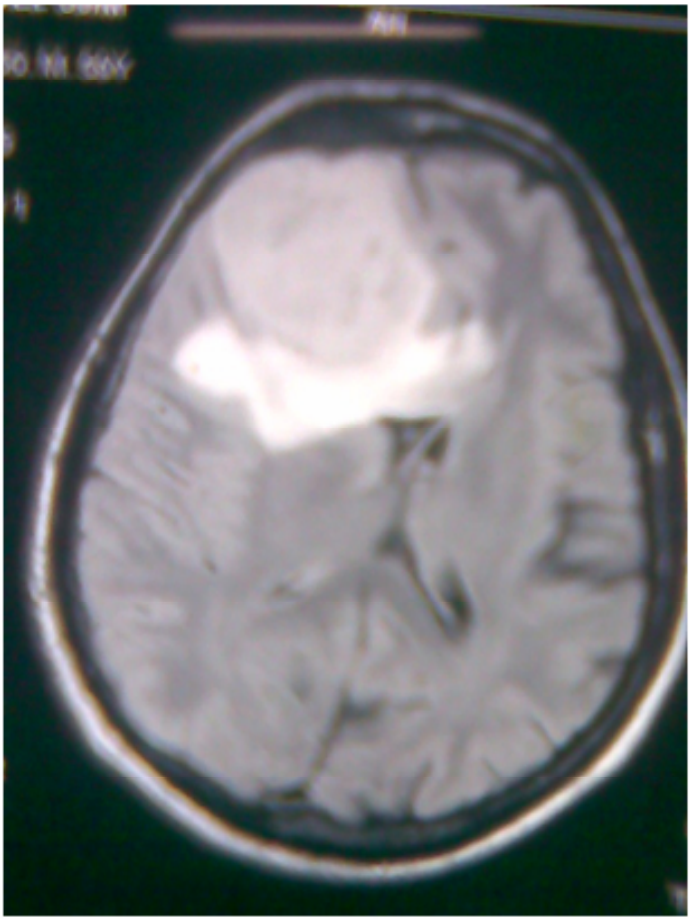


Table 13:Basisc underlying cause in patients who succumbed

Etiology	No
Endosulfan poisoning	1
Hanging with hypoxic encephalopathy	1
CVA –massive infarct	2
SOL-primary	1
CAD/ACS/acute ICH	1
Bronchogenic carcinoma with secondaries	1
DM /hydronephrosis	1
Metobolic encephalopathy/refractory seizure	1

Mortality is associated with the underlying etiology and not altered by occurrence of single episode of seizure.Tumors,end stage renal disease,large hemorrhage,large infarct,severe hypoxia ,drug withdrawal complicated infection, all have poor prognosis (Table 13).

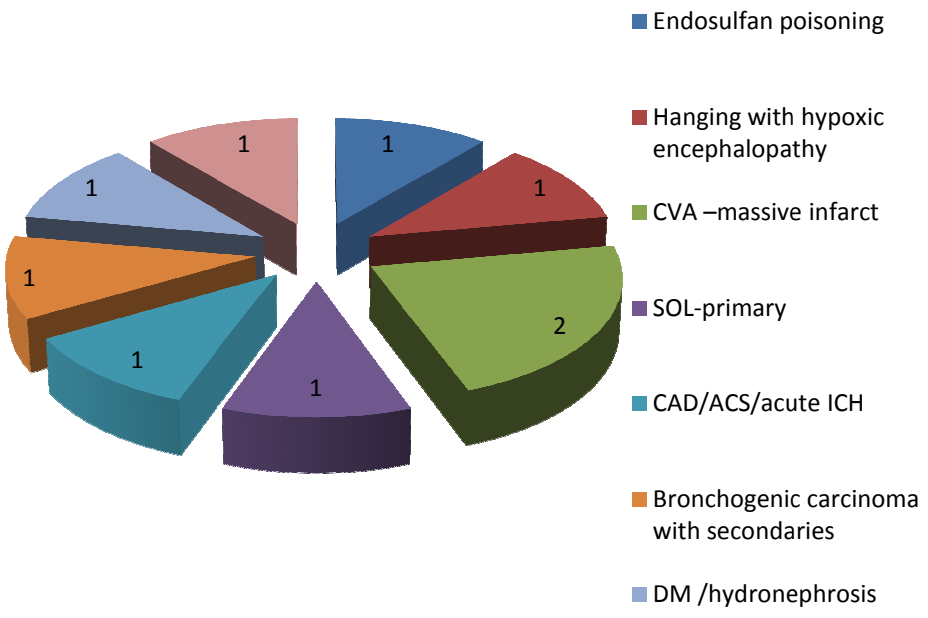


Table 14:

Sex wise distribution of mortality

Sex	Died		Survived		Total		Ψ^2	d.f.	Significance
	fre	%	fre	%	fre	%			
Male	5	55.6	34	66.7	39	65.0	0.415	1	P=0.519
Female	4	44.4	17	33.3	21	35.0			
Total	9	100	51	100	60	100			

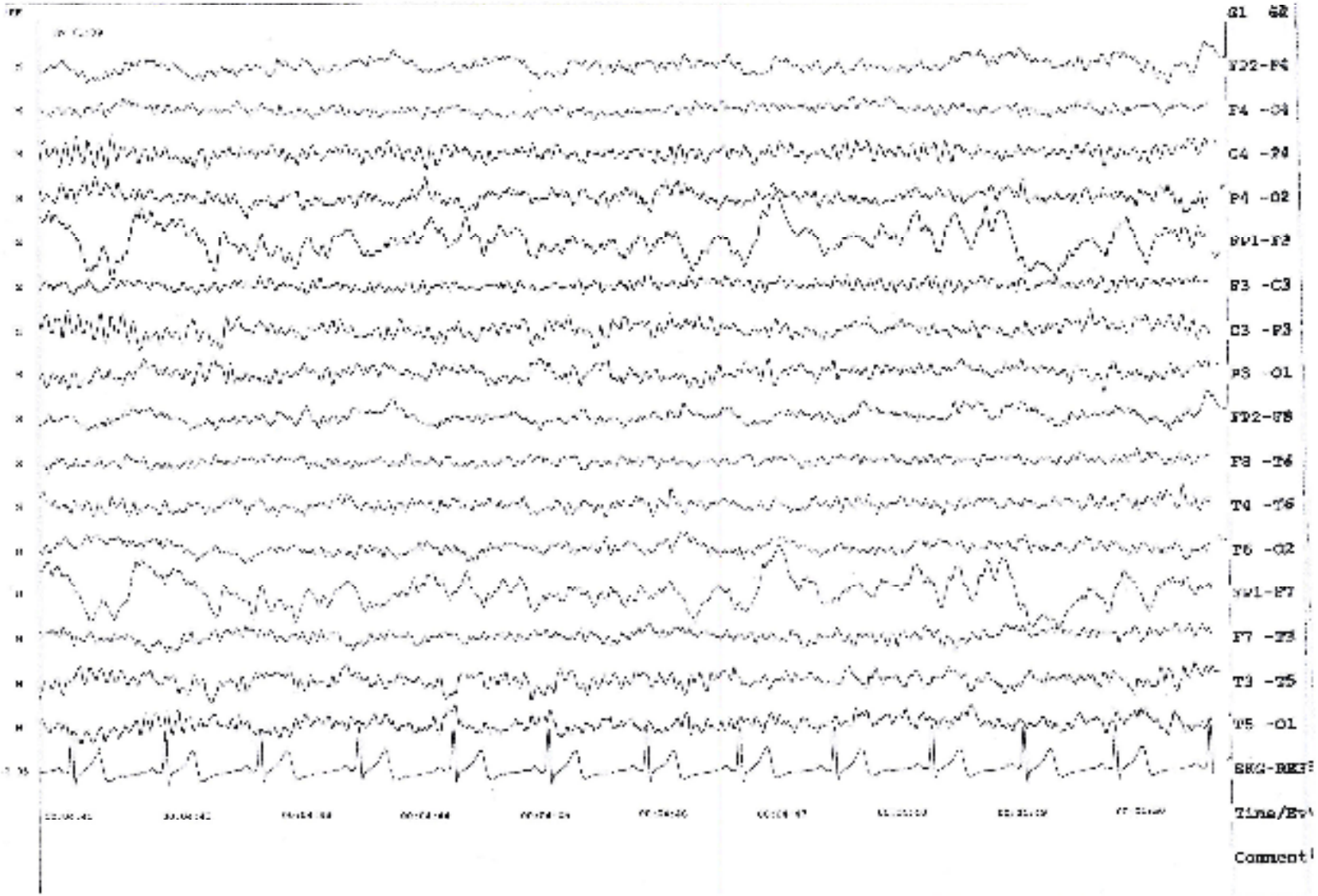
The above table – 15 reveals the association of mortality with the sex of the patients. Among the total 9 mortality cases, 5(55.6%) cases were male and the remaining 4(44.4%) cases were females. The sex wise distribution of mortality is not statistically Significant (P>0.05).

Table 15: Association of mortality with status and underlying cause

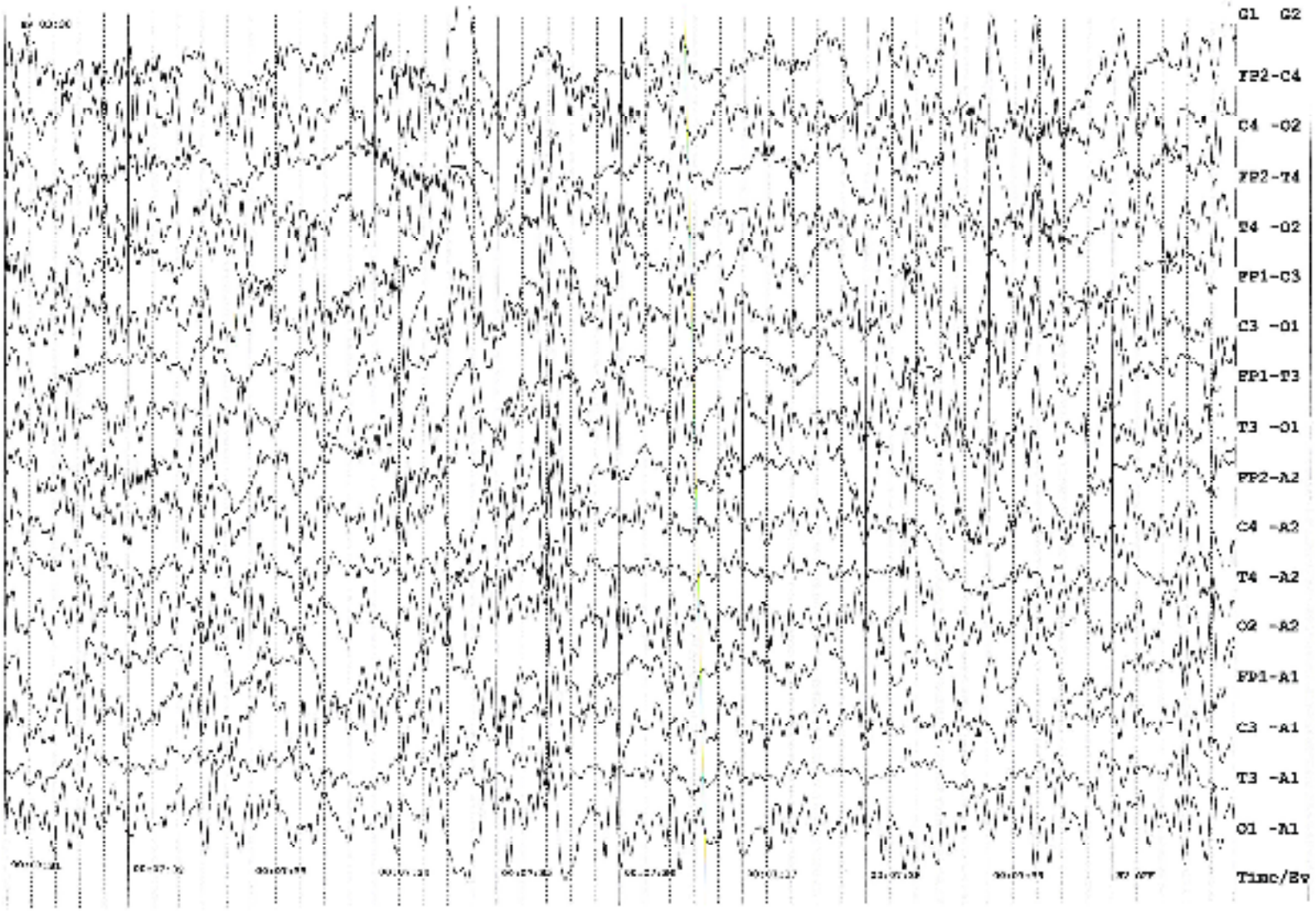
Cause	Status YES/NO	NO
Endosulfan poisoning	YES	1
Hanging with hypoxic encephalopathy	YES	1
CVA –massive infarct	YES	2
SOL-primary	NO	1
CAD/ACS/acute ICH	NO	1
Bronchogenic carcinoma with secondaries	NO	1
DM /hydronephrosis	YES	1
Metabolic encephalopathy/refractory seizure	NO	1

Though the cause for status epilepticus varies among the study group, mortality depends upon the severity of the underlying disease as shown in table 15.

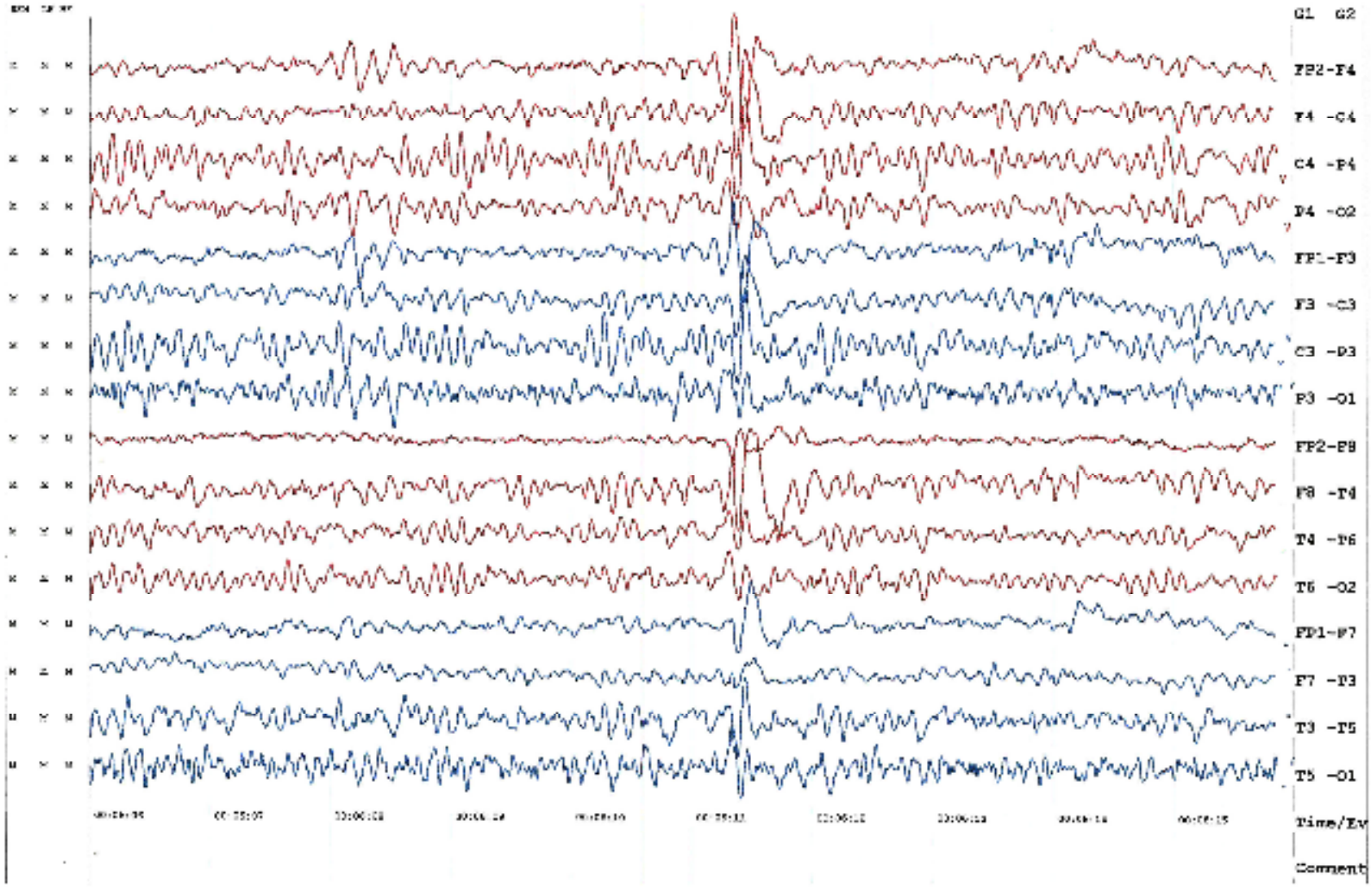
Normal EEG (Patient No : 8)



EEG showing Background α rthythm with bilateral abnormal epileptiform discharges (Patient No : 14)



EEG showing abnormal epileptiform discharges



DISCUSSION

Among the total study subjects(60),the number of males were 39(65%),and the females were 21(35%)(table-1) Most authors report a small-to-moderate preponderance of men in their studies of first seizures in adults (van Donselaar20, 1992; Musicco, 1997;Hopkins18, 1988; King, 1998). A male to female ratio of **1.8: 1** is observed in this study, a trend noted in other studies.

The age of study population ranges from 13-89 years. The mean age of onset of seizures for the males was 43.9±20,for the females was 33.3±16.2 years. The occurrence of seizures is earlier in females than the males since the mean age for the females was statistically lower than the mean age for the males ($P<0.05$)(Table-2)

On analyzing the past history of seizures,24(40%) cases had past history of seizure and the remaining 36(60%) had new onset seizure. The mean age for the known seizure patients is 37.3±16.3,and for the new onset cases 42.5±21.(Table-4)

The etiology for the occurrence of seizure varies in the known seizure disorder patients, most of them had seizures due to the drug withdrawal (83.3%).about 12.5% cases had vascular etiology, 4.2%cases had infection as the cause(Table-5)

The etiology for the recurrence of seizure episode in known epileptic varies.The most common cause being noncompliance of drugs.

In this study two patients had acute parenchymal hemorrhage induced seizure. One patient had hyperglycemia induced seizure which presented as Epilepsia Partialis Continua.(Table-5)

The dominant causes for the development of new onset seizure were vascular(25%) and metabolic abnormalities(25%) .Hyperglycemia is the most frequent metabolic abnormality encountered. Others are hypoglycemia, hyponatremia, and uremia.(Table-6)

Hypoxia induced seizure had occurred in 4 patients. Attempted partial hanging with hypoxia resulted in GTCS type of seizure in 3 patients.

The seizure type classified in this study as per International League Against Epilepsy-revised classification of epileptic seizures revealed generalized seizure (including secondary generalization) in 83.3% and partial seizure in 16.7 %.(table -7)

Zhu PG studied new onset seizures in the ages between 20 and 80 revealed generalized seizures in 64% and partial in 30%.

Retrospective study of Perez et al in 250 patients with late onset seizures revealed 59% generalized and 41% partial in nature.

The observation made by Bleck TP,smithMC,Piere Louis SJC et al revealed that most seizures occurring in the ICU setting manifest as generalized tonic clonic convulsions (90%)including secondary generalization.

The observation of seizure type in IMCU in this study is almost similar to the study by Bleck et al[14,15].

In contrary, a recent study of Prego-*Lopez M, Devinsky O identified* partial seizures as the most common seizure type in adults.

Table 16.compares seizure type encountered in this study with various studies.

Seizure type	This study	Bleck et al	Zhu PG
GTCS	83.3%	90%	64%
Partial	16.7%	10%	30%

The seizure typing in this study was entirely made with history and observation, .It may be considerably difficult to diagnose patients who present with complex partial and other non convulsive seizure, especially in a critical care setting where sedative medications are often administered but such patients are uncommon

On analyzing the sex wise distribution of seizure pattern, GTCS was the most common seizure type in both sexes. 23 (59%) males and 15 (71.4%) females were with GTCS type of seizure.

The type of seizure was analyzed according to the age of the patient .GTCS type of seizure was the most common type of seizure in both less than median age group and population at and above the median age.(table-8)

The incidence of status epilepticus in this study was 31.67%(19 patients).Drug withdrawal is the most common cause for the status epilepticus and accounts for 6 out of 19 cases of status epilepticus.

Among the status epilepticus patients generalized convulsive status epilepticus is seen in 15(78.9%) of cases and partial status epilepticus was seen in 4(21.1%) cases.

In patients with no previous history of seizure, the common abnormality associated with status epilepticus is hyperglycemia. All the three patients with hyperglycemia induced seizure presented with Epileptia Partialis continua. Hypoglycemia presented as generalized convulsive status epilepticus(GCSE).Most other etiologies had GCSE.

Mortality associated with status was seen in 4 cases, among which underlying etiology was irreversible in 3 patients which resulted in death. By the appropriate use of status epileptic treatment protocol (Table-A,B,C), status epilepticus in all patients were in good control. Hyperglycemia was treated with standard insulin regimen and cerebral edema was relieved with anti edema measures.

The common CT abnormality was the chronic infarct with gliosis. Some showed tumors,parenchymal haemorrhage,ring enhancing lesion and calcification (table-12)

De Lorenzo & his colleagues in the prospective population based epidemiological study in Richmond,Virginia found that partial status

epilepticus of various types occur in 25% of cases &non convulsive status epilepticus in about 4% of status epilepticus.

Table -17 Comparison of types of status epilepticus

Type of STE	This study	De Lorenzo & colleagues[18]
GCSE	78.9%	61%
Partial SE	21.1%	25%

Only six (31.5%) of the 19 cases of status epilepticus had past history of seizures,and the remaining 13 (68.5%) cases ha new onset seizure with status .Among the total 36 new onset cases ,13 (36.1%) cases had status epilepticus.

In the retrospective study from Mayo clinic,Wijidicks and sharbrough reported that only 7.3% of new onset seizure cases had status epilepticus.[15] .

This study has a higher incidence of status epilepticus in the new onset seizure group compared to the above study.

The largest single identifiable factor for the GCSE is epilepsies associated with low levels of antiepileptic drugs(AEDs);42%in the Richmond study had epilepsy and 34% of adults had low AED levels .

Cerebro vascular diseases and discontinuation of drugs were the most prominent causes of status epilepticus in another study from Richmond,Virginia,each accounting for 22% of all in patient series[18] .

Out of the nine mortality cases 4(21.6%) cases had status epilepticus .the mortality rate in various studies for GCSE ranges from 15-22% in adults [18,19,20,21]

The study conducted by S. K. Gupta, Ashok Parihar in GMC, Jammu &Kasmir revealed that the metabolic abnormalities are responsible for the up to 30-35% of seizures in critically ill patients[22].

CONCLUSIONS

1. The incidence and prevalence of seizure in IMCU was slightly higher in males than in females
2. Mean age of onset seizure for males was 43.9±2, and for females was 33.3±16.2 indicating that more females were admitted at younger age with seizure in this study
3. Drug withdrawal is the commonest cause of recurrence of seizure in known epileptics
4. Vascular and metabolic abnormalities are the dominant causes for new onset seizure in IMCU with equal frequency
5. Hyperglycemia was the commonest metabolic abnormality encountered which presented as Epileptia partialis continua
6. Most patients had GTCS type of seizure(including secondary generalization) ,as seen in 85% of patients
7. The age of the patient does not influence the type of seizure
8. Status epilepticus was mainly due to metabolic abnormality in new onset seizure patients
9. Nature of the critical illness determines the outcome, not altered by occurrence of single episode of seizure or even status epileptcus
10. Use of specialized treatment protocol for treatment of IMCU seizure had resulted in good recovery of the study subjects

SUMMARY

Even though the etiology and presentation of seizure in critically ill patients vary, the most common reversible causes like metabolic abnormalities, infections, toxins should be sought out in all the cases; and by the appropriate management of underlying critical illnesses and by the use of specialized treatment protocol for the IMCU seizures, good recovery is possible.

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PROFORMA

NAME: AGE:

SEX:

Occupation: IP no. Date and time of admission:

Date and time of onset of seizure:

Type of seizure:

GTCS	SPS	CPS	SPGT	CPGT	Myoclonus	Others

Frequency of seizures-

Whether known case of seizure- Yes/No

If yes, H/o discontinuation of drugs – Yes/No

H/o fever, vomiting, Headache- Yes/No

H/o fever, altered sensorium- Yes/No

H/o trauma- Yes/No

H/o alcohol ingestion- Yes/No

H/o any hypoxic state- Yes/No

H/o poisoning/ drug overdose- Yes/No

If yes what drug-

H/o suggestive of CVA- Yes/No

H/o suggestive of SOL- Yes/No

H/o suggestive of metabolic abnormality- Yes/No

PAST H/o:

H/o DM/HT/CAD/ BA/COPD/PT/HIV

Examination:

Vitals PR- BP- RR- Temperature-

GLASGOW COMA SCORE			
Eye Opening	**Verbal (Nonintubated)**	**Verbal (Intubated)**	**Motor Activity**
4—Spontaneous	5—Oriented and talks	5—Seems able to talk	6—Verbal command
3—Verbal stimuli	4—Disoriented and talks	3—Questionable ability to talk	5—Localizes to pain
2—Painful stimuli	3—Inappropriate words	1—Generally unresponsive	4—Withdraws to pain
1—No response	2—Incomprehensible sounds		3—Decorticate
	1—No response		2—Decerebrate
			1—No response

Fundus-

Cranial nerves-

Motor system-

Sensory system-

Spine and skull-

Meningeal signs-

Other systems-

CVS-

RS-

P/A-

Investigations:

1. Urine Alb- sugar- depostis- ketones-
2. TC DC Hb ESR
3. Blood sugar urea creatinine
4. Serum Na+ K+ Ca++
5. LFT Bn SGOT SGPT SAP S.protein S.Albumin
6. HIV ELISA-
7. Lumbar puncture
8. ABG
9. ECG
10. Chest Xray
11. CT scan
12. MRI
13. EEG
14. Others-Outcome

S.No.	Age	Sex	IPno.	Known SD	Etiol if yes	New onset	type	STE	NCS	NS	CT	MRI	EEG	Outcome	Eitology
1	50	M	24551	Y	VAS	N	SPGT	N	Y	Y	A	A	–	D	Vas
2	34	M	24876	Y	DW	N	GTCS	N	N	N	Nr	–	Nr	D	DW
3	45	F	25443	Y	DW	N	GTCS	N	Y	N	Nr	–	–	D	DW
4	75	M	14277	N		Y	SPGT	N	N	N	A	A	–	D	TU
5	60	M	25711	N		Y	SPGT	N	N	Y	Nr			D	Met
6	29	M	35983	Y	DW	N	GTCS	N	N	N	A	–	–	D	DW
7	20	M	27765	Y	DW	N	SPS	N	N	N	Nr	–	A	D	DW
8	20	M	27001	Y	DW	N	CPS	N	N	N	Nr	–	Nr	D	DW
9	47	F	28090	N		Y	SPS	Y	Y	N	Nr	–	A	D	TUM
10	46	M	41473	N		Y	GTCS	Y	Y	N	Nr	–	–	D	Tox
11	44	M	41412	N		Y	GTCS	Y	Y	N	Nr	–	–	D	Tox
12	80	M	20607	N		Y	SPS	Y	Y	Y	Nr	–	–	D	Met
13	86	M	28077	N		Y	SPS	Y	Y	Y	Nr	–	–	D	Met
14	13	M	7180	Y	DW	N	GTCS	Y	N	N	Nr	–	A	D	DW
15	20	F	42393	N		Y	GTCS	Y	N	N	N	–	–	E	Tox
16	20	F	42313	N		Y	GTCS	Y	Y	Y	Nr	–	–	E	Hyp
17	39	M	41836	Y	DW	N	GTCS	N	Y	N	A	–	–	D	DW
18	27	F	42259	N		Y	GTCS	Y	N	N	Nr	–	–	D	Hyp
19	17	F	42111	N		Y	GTCS	Y	Y	N	Nr	–	–	D	HYP
20	53	M	46687	Y	DW	N	CPS	N	N	N	A	–	–	D	DW
21	13	M	41742	Y	DW	N	GTCS	N	Y	N	Nr			D	DW
22	67	M	41819	Y	VAS	N	GTCS	N	N	Y	Nr	–	–	D	VAS
23	56	M	43891	Y	DW	N	GTCS	N	Y	Y	A	–	–	D	DW
24	53	M	43901	N		Y	CPS	N	Y	Y	A	–	–	E	Vas
25	34	F	43674	N		Y	SPS	Y	N	N	Nr	–	–	D	Met
26	13	F	44378	N		Y	GTCS	N	Y	N	Nr	–	–	D	UNI
27	48	F	44341	Y	DW	N	GTCS	N	Y	N	Nr	–	–	D	DW
28	17	F	43970	Y	Met	N	GTCS	N	N	N	Nr	–	–	D	Met
29	23	F	40508	N		Y	GTCS	N	Y	Y	A	A	–	D	SOL
30	28	F	43423	Y	DW	N	myoclo	N	Y	N	Nr	–	A	D	DW
31	27	F	41037	N		Y	CPGT	N	Y	Y	Nr	A	–	D	INF
32	30	M	43758	N		Y	GTCS	N	Y	Y	A	A	–	F	Trm
33	36	M	44193	Y	Vas	N	GTCS	N	Y	Y	A	–	–	E	Vas
34	33	M	43509	N		Y	CPGT	Y	Y	Y	A	–	–	D	Vas
35	60	M	44553	Y	DW	N	CPGT	Y	Y	Y	A	–	–	D	DW
36	21	M	43978	N		Y	GTCS	N	Y	Y	A	–	–	D	INF
37	41	M	42867	N		Y	CPGT	N	Y	Y	A	–	–	E	Tum
38	45	F	41374	Y	DW	N	GTCS	N	N	N	Nr	–	–	D	DW
39	50	M	44867	Y	DW	N	GTCS	Y	N	Y	A	–	–	E	DW
40	70	M	44633	N		Y	SPS	N	N	Y	A	–	–	D	Vas
41	65	F	44987	Y	DW	N	GTCS	N	N	N	Nr	–	–	D	DW
42	42	M	28143	N		Y	GTCS	N	Y	N	Nr	–	–	D	Met
43	65	F	45271	N		Y	GTCS	Y	Y	Y	Nr	–	–	E	Met
44	27	M	45280	N		Y	CPGT	Y	Y	Y	A	A	–	D	Vas
45	21	F	44983	N		Y	GTCS	N	N	N	Nr	–	–	D	Hyp
46	58	F	47161	N		Y	GTCS	N	Y	N	Nr			D	Met
47	28	F	47121	N		Y	GTCS	Y	Y	N	Nr	–	–	D	INF
48	45	M	46749	N		Y	GTCS	N	Y	Y	Nr	–	–	F	Met
49	30	M	46997	N		Y	GTCS	N	Y	Y	Nr	Nr	–	D	UNI
50	56	M	45997	N		Y	GTCS	N	Y	Y	A	–	–	D	Vas
51	56	F	25133	N		Y	GTCS	N	Y	Y	Nr	–	–	D	Met
52	40	M	23936	Y	DW	N	CPGT	N	Y	Y	A	A	–	D	Vas
53	46	M	26230	N		Y	GTCS	N	Y	N	A	–	–	D	Vas
54	17	M	26306	Y	DW	N	GTCS	Y	N	N	Nr	–	–	D	DW
55	36	M	27123	Y	DW	N	SPGT	N	Y	Y	A	–	–	D	VAS
56	25	M	27608	N		Y	GTCS	N	Y	N	Nr	–	–	D	INF
57	27	M	27828	N		Y	GTCS	N	N	Y	A	–	–	D	Vas
58	51	M	28451	N		Y	SPGT	N	N	N	A	–	–	D	Vas
59	22	F	28503	Y	DW	N	SPGT	Y	N	N	Nr	–	–	D	DW
60	69	M	29535	N		Y	GTCS	N	Y	Y	A	–	–	D	Vas

KEY TO THE MASTER CHART

A	→	Abnormal
CPGT	→	Complex partial with secondary generalization
CPS	→	Complex partial seizure
CT	→	CT Scan
D	→	Discharge
DW	→	Drug withdrawal
E	→	Expired
F	→	Female
GTCS	→	Generalised tonic clonic seizures
Hyp	→	Hypoxia
INF	→	Infection
M	→	Male
MET	→	Metabolic cause
MRI	→	Magnetic Resonance Imaging
Myoclo	→	Myoclonus
N	→	No
Nr	→	Normal
NS	→	Neurological signs on admission
SD	→	Seizure Disorder
SOL	→	Space occupying lesion
SPGT	→	Simple partial with secondary generalization

SPS	→	Simple partial seizure
STE/SE	→	Status Epileticus
Tox	→	Toxin
Tum	→	Tumor
UNI	→	Unidentified
VAS	→	Vascular
Y	→	Yes